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Dilated Cardiomyopathy and Severe Functional Valvular Regurgitation in a Young Adult with Prior Childhood Chemotherapy: Progressive Ventricular Dysfunction Despite Surgical Correction and Optimal Medical Therapy

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ABSTRACT
Background: Late cardiotoxicity is a recognized complication in survivors of childhood cancer treated with potentially cardiotoxic agents, particularly anthracyclines. These therapies may cause progressive myocardial injury through mechanisms involving oxidative stress, mitochondrial dysfunction, and cardiomyocyte apoptosis. Over time, this process can lead to ventricular remodeling, dilated cardiomyopathy, and heart failure. Advanced ventricular dilation frequently results in functional mitral and tricuspid regurgitation, further worsening hemodynamic burden and clinical outcomes.
Case summary: We report the case of a 42-year-old man with a history of non-Hodgkin lymphoma diagnosed in childhood and treated with chemotherapy, without subsequent cardiologic follow-up. From the age of 29, he developed progressive heart failure symptoms. Transthoracic echocardiography revealed severe functional mitral regurgitation with mixed mechanism (Carpentier type I due to annular dilation and type IIIa due to leaflet restriction) and massive tricuspid regurgitation, associated with severe atrial dilation, grade III diastolic dysfunction, and pulmonary hypertension. Left ventricular systolic dysfunction was documented with an ejection fraction of 36% and markedly reduced global longitudinal strain (−11%), consistent with non-ischemic dilated cardiomyopathy.
The patient underwent mitral valve replacement and tricuspid valve repair but developed intraoperative cardiac arrest and postoperative mixed shock. Persistent severe ventricular dysfunction (LVEF 26%, GLS −8%) and multiple complications were observed.
Conclusion: This case highlights the importance of long-term cardiovascular surveillance in childhood cancer survivors and the role of strain imaging in detecting chemotherapy-related cardiomyopathy.

KEYWORDS: Dilated cardiomyopathy, Cardio-oncology, Anthracycline cardiotoxicity, Mitral regurgitation, Tricuspid regurgitation, Global longitudinal strain, Childhood cancer survivors, Heart failure

Advances in oncologic therapies have dramatically improved survival among children diagnosed with cancer, with current survival rates exceeding 80% in developed countries. However, this improvement has led to increasing recognition of long-term treatment-related complications, particularly cardiovascular disease, which has emerged as a leading cause of late morbidity and mortality among cancer survivors [1]. Anthracycline-based chemotherapy, widely used in the treatment of hematologic malignancies such as non-Hodgkin lymphoma, is strongly associated with dose-dependent cardiotoxicity that may manifest years or even decades after exposure [2].
Anthracycline-induced cardiotoxicity results from complex mechanisms including oxidative stress, mitochondrial dysfunction, topoisomerase IIβ–mediated DNA damage, and activation of apoptotic pathways within cardiomyocytes. These processes ultimately lead to progressive myocardial cell loss, ventricular remodeling, and the development of dilated cardiomyopathy[3]. Importantly, the clinical course may remain asymptomatic for prolonged periods before progressing to overt heart failure with reduced ejection fraction.
Long-term survivors of childhood cancer are known to have a substantially increased lifetime risk of developing heart failure, cardiomyopathy, and other cardiovascular complications compared with age-matched controls. Population-based studies have shown

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that the cumulative incidence of heart failure among survivors treated with cardiotoxic therapies may reach 4–10% by mid-adulthood, with significantly higher risks in those exposed to high cumulative doses of anthracyclines or chest radiation [4].

In advanced stages of dilated cardiomyopathy, ventricular remodeling frequently leads to secondary functional valvular regurgitation, particularly involving the mitral and tricuspid valves. These lesions arise from ventricular dilation, papillary muscle displacement, and annular enlargement rather than intrinsic leaflet pathology. Severe functional regurgitation further worsens ventricular loading conditions, contributing to a vicious cycle of progressive ventricular dysfunction and heart failure progression [5].

Echocardiographic myocardial deformation imaging, particularly global longitudinal strain (GLS), has emerged as a sensitive marker for the early detection of myocardial injury in cardiooncology populations. GLS abnormalities often precede measurable declines in left ventricular ejection fraction and have been shown to provide prognostic information regarding future cardiac dysfunction [6].

In this context, we report the case of a young adult with a history of childhood chemotherapy who developed non-ischemic dilated cardiomyopathy complicated by severe functional mitral and tricuspid regurgitation, with progressive clinical deterioration despite surgical correction and optimal medical therapy.

We report the case of a 42-year-old man with a history of non-Hodgkin lymphoma diagnosed during childhood and treated with chemotherapy, without subsequent structured cardiologic follow-up. Beginning at the age of 29, he developed progressive symptoms of heart failure characterized by exertional dyspnea and gradual functional deterioration.

Echocardiographic evaluation performed in 2022 demonstrated severe functional mitral regurgitation classified as Carpentier type V and massive tricuspid regurgitation, associated with severe atrial dilation, grade III diastolic dysfunction, and signs of pulmonary hypertension. The left ventricle exhibited systolic dysfunction with an ejection fraction of 36%, together with markedly impaired myocardial deformation with a global longitudinal strain of −11%, findings consistent with advanced non-ischemic dilated cardiomyopathy.

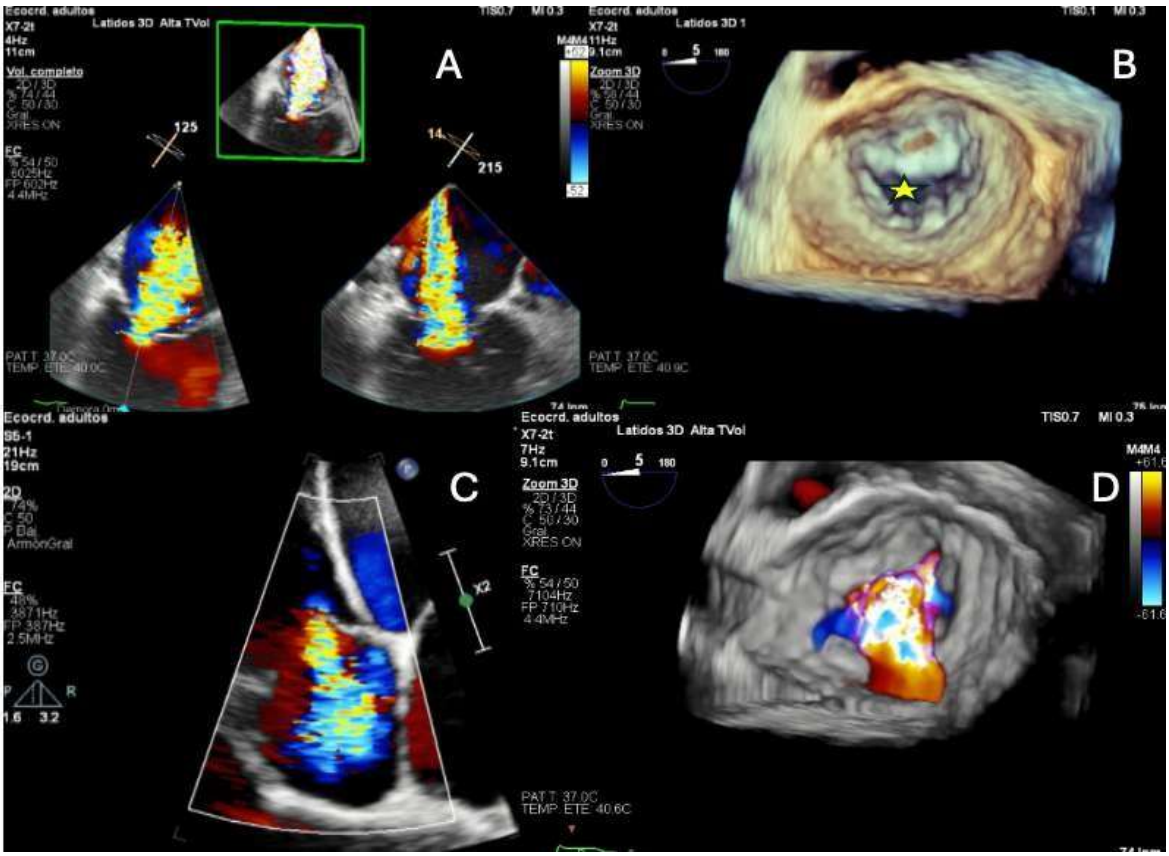

Figure 1. Echocardiographic assessment of severe atrioventricular valve regurgitation.

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(A) Three-dimensional echocardiographic view of the mitral valve demonstrating a severe mitral regurgitation jet. (B) Three-dimensional en face view of the mitral valve showing an eccentric regurgitant orifice*, consistent with mixed-mechanism mitral regurgitation: Carpentier type I (atrial annular dilation) and type IIIa (leaflet restriction). (C) Two-dimensional color Doppler image demonstrating severe tricuspid regurgitation. (D) En face color Doppler view of the mitral valve demonstrating multiple confluent mitral regurgitation jets.

Given progressive clinical deterioration and the severity of valvular regurgitation, the patient underwent cardiac surgery consisting of mitral valve replacement with a mechanical prosthesis and tricuspid valve repair using a Cosgrove annuloplasty ring. During the procedure, the patient developed intraoperative cardiac arrest requiring prolonged cardiopulmonary resuscitation and advanced circulatory support.

In the immediate postoperative period, the patient developed post-cardiac arrest syndrome and mixed shock with cardiogenic, vasoplegic, and septic components. Severe left ventricular systolic dysfunction persisted with an ejection fraction of 26%, accompanied by further deterioration of myocardial deformation with an estimated GLS of −8%, suggesting progression of the underlying myocardial injury.

The hospital course was further complicated by subarachnoid hemorrhage, acute kidney injury stage KDIGO II, and nosocomial sepsis secondary to ventilator-associated pneumonia and complicated urinary tract infection. The patient required prolonged mechanical ventilation, multiple vasopressors, and broad-spectrum antibiotic therapy.

Figure 2. Advanced echocardiographic findings demonstrating severe ventricular dysfunction and persistent valvular regurgitation.

(A) Left ventricular global longitudinal strain (GLS) bull's-eye plot demonstrating severely reduced strain across all myocardial segments, consistent with non-ischemic dilated cardiomyopathy. (B) Increase in tricuspid regurgitation jet following surgical intervention. (C) Reduced left ventricular ejection fraction (LVEF) demonstrating absence of reverse remodeling. (D) Peak tricuspid regurgitation velocity, suggesting high probability of pulmonary hypertension.

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This case illustrates the complex clinical course that may occur in patients with advanced cardiomyopathy related to late chemotherapy-associated cardiotoxicity. Despite appropriate surgical correction of severe functional valvular regurgitation and optimal guideline-directed medical therapy for heart failure, persistent deterioration of ventricular function suggests irreversible myocardial injury.

Furthermore, this case highlights the clinical importance of early cardiovascular surveillance in childhood cancer survivors. Current cardio-oncology guidelines emphasize the need for lifelong monitoring in patients exposed to cardiotoxic therapies, particularly those treated with anthracyclines, in order to identify subclinical myocardial dysfunction and initiate cardioprotective strategies at earlier stages of disease progression.

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